CLAIMS

1. An isolated nucleic acid comprising at least 63 contiguous nucleotides of SEQ ID NO:1 or the complement thereof.

2. The isolated nucleic acid of claim 1, wherein the isolated nucleic acid is an expression vector.

3. The isolated nucleic acid of claim 2, wherein the expression vector is a viral vector.

4. The isolated nucleic acid of claim 3, wherein the viral vector is an adenoviral vector.

5. An isolated nucleic acid comprising a nucleic acid sequence that encodes a transdominant negative hKIS protein, polypeptide or peptide.

6. The isolated nucleic acid of claim 5, wherein the transdominant negative hKIS protein, polypeptide, or peptide contains a mutation altering its serine/threonine kinase activity.

7. The isolated nucleic acid of claim 6 comprising SEQ ID NO: 3 or a functional equivalent thereof.

8. An isolated nucleic acid encoding a cyclin kinase inhibitor containing a mutation at a serine or threonine amino acid, wherein the cyclin kinase inhibitor retains its ability to arrest cells in G1 phase.

9. The isolated nucleic acid of claim 8, wherein the cyclin dependent kinase inhibitor is p16, p21, p27, or p57.

10. The isolated nucleic acid of claim 9, wherein the cyclin dependent kinase inhibitor is p27.

11. The isolated nucleic acid of claim 10, wherein the mutation encodes a change of a serine codon to encode another amino acid.

12. The isolated nucleic acid of claim 11, wherein the serine codon corresponds to amino acid 10 of wild-type p27.

13. The isolated nucleic acid of claim 12, wherein the serine codon is mutated to encode alanine.

14. A polypeptide, encoded within an expression vector, comprising a cyclin kinase inhibitor containing a mutation at a serine or threonine amino acid, wherein the cyclin kinase inhibitor retains its ability to arrest cells in G1 phase.

15. A kit comprising an isolated nucleic acid of claim 5.

16. A kit comprising an isolated nucleic acid of claim 8.

17. A method of inhibiting proliferation of a cell comprising the step of contacting the cell with a nucleic acid encoding a CKI protein that is not functionally inhibited by a serine/threonine kinase.

18. The method of claim 17, wherein the cell is a mammalian cell.

19. The method of claim 18, wherein the mammalian cell is a human cell.

20. The method of claim 17, wherein the mammalian cell is a neointimal cell.

21. The method of claim 17, wherein the CKI protein comprises a mutation in a serine/threonine kinase binding domain of the protein.

22. The method of claim 17, the CKI protein comprises a mutation in a domain phosphorylated by a serine/threonine kinase.

23. The method of claim 17, wherein the CKI protein is p27.

24. The method of claim 17, wherein the mutation encodes a change of a serine codon to encode another amino acid.

25. The method of claim 24, wherein the other amino acid is alanine.

26. The method of claim 24, wherein in the serine residue corresponds to amino acid 10 of wild-type p27.

27. The method of claim 17, wherein the isolated nucleic acid is an expression vector.

28. The method of claim 27, wherein the expression vector is a plasmid.

29. The method of claim 27, wherein the expression vector is a viral vector.

30. A method of inhibiting the proliferation of a cell comprising contacting the cell with a composition inhibiting phosphorylation of p27 by hKIS.

31. A method of treating a patient comprising administering an isolated nucleic acid encoding a CKI protein that is not functionally inhibited by a serine/threonine kinase.

32. A method of treating or otherwise ameliorating a cell proliferative disorder in a patient comprising the step of providing to a proliferating cell to be treated a cyclin dependent kinase modified at a serine or threonine residue such that the cyclin dependent kinase is not phosphorylable by a serine/threonine kinase.

33. A kit comprising a catheter and a solution comprising an isolated nucleic acid encoding a cyclin kinase inhibitor containing a mutation at a serine or threonine codon, wherein the cyclin kinase inhibitor retains its ability to arrest cells in G1 phase.